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**ORIGINAL ARTICLE**

# Increased eating frequency linked to decreased obesity and improved metabolic outcomes

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**BACKGROUND:** We previously reported that more frequent eating in overweight minority youth was linked to lower visceral adiposity and circulating triglycerides. The aim of this study was to examine this issue in more detail by assessing the relationship between eating frequency and adiposity and metabolic disease risk in a cohort of exclusively overweight Hispanic youth.
**METHODS:** This analysis included 191 overweight (⩾85th percentile body mass index (BMI)) Hispanic youth (8–18 years) with the following cross-sectional measures: height, weight, BMI, dietary intake via multiple 24 h recalls, body composition via dual-energy X-ray absorptiometry, lipids and insulin action (insulin sensitivity, acute insulin response (AIR) and disposition index, a measure of β-cell function) via a frequently sampled intravenous glucose tolerance test. Each eating occasion (EO) was defined as ⩾50 calories and ⩾15 min from any prior EO. Infrequent eaters (IEs) were classified as any subject who ate $<3$ EOs on any dietary recall ($n=32$), whereas frequent eaters (FEs) always consumed ⩾3 EOs ($n=159$).
**RESULTS:** Using analyses of covariance, FEs compared with IEs consumed 23% more calories per day ($P\leqslant0.01$), ate 40% more often and consumed 19% less calories per EO ($P\leqslant0.01$). FEs also exhibited 9% lower BMI *Z*-scores ($P\leqslant0.01$), 9% lower waist circumferences ($P\leqslant0.01$), 29% lower fasting insulin ($P=0.02$), 31% lower HOMA-IR (Homeostatic Model Assessment: Insulin Resistance) values ($P=0.02$) and 19% lower triglycerides ($P\leqslant0.01$), as well as an 11% higher AIR ($P=0.02$) and 31% higher disposition index ($P=0.01$). The following *a priori* covariates were included: Tanner, sex, body fat and reported energy intake.
**CONCLUSION:** These findings suggest that increased eating frequency is related to decreased obesity and metabolic disease risk in overweight Hispanic youth, despite increases in energy intake.

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## INTRODUCTION

Hispanics are the largest and fastest growing ethnic minority in the United States, representing 17% of the population.[1] Obesity and type 2 diabetes also disproportionately affect Hispanics. National data collected in Hispanic adolescents (12–19 years) show that 42% are overweight and 23% are obese, compared with 30% and 16%, respectively, in non-Hispanic Whites (NHW).[2] Furthermore, our group has shown that over 30% of Hispanic children and adolescents (8–18 years) have prediabetes and the metabolic syndrome.[3,4]

Hispanic youth are also more likely to eat less often than non-Hispanic Whites,[5] and national data suggest that approximately 9% of Hispanic adults compared with 3% of non-Hispanic Whites consume ⩽2 eating occasions (EOs) per day.[6] Yet, it remains unclear how or if this decreased number of EOs per day affects obesity and metabolic diseases within this high-risk population. Our group has recently shown in a combined sample of overweight African-American and Hispanic youth (8–18 years) that frequent eating (⩾3 EOs per day) compared with infrequent eating ($<3$ EOs per day) was associated with decreased visceral adiposity and triglycerides, despite being linked to increased reported daily energy intake.[7] The aim of this study is to expand on our previous research using the same EO definition[8] and examine these relationships in a younger and more diverse Tanner-staged sample of exclusively overweight Hispanic Youth, while also broadening the scope of our dietary analysis by examining the percentage of calories eaten in the morning, afternoon and evening based on previous work by Thompson *et al.*[9]

## MATERIALS AND METHODS

### Subjects

The design, data collection procedures and findings of the University of Southern California longitudinal SOLAR (Study of Latino Adolescents at Risk for Diabetes) cohort, which began in the year 2000, have been described in detail previously.[10,11] Although cross-sectional dietary analyses have previously been conducted in this cohort,[12,13] the present analysis is the first to examine eating frequency and adiposity, as well as metabolic disease risk using this cohort. One hundred and ninety-one participants, for whom both complete body composition and dietary data were available, were included in this analysis. One subject did not have a 2-h oral glucose tolerance test (OGTT) and smaller subsamples had specific metabolic measures from a frequently sampled intravenous glucose tolerance test (FSIVGTT). Inclusion criteria for SOLAR were as follows: (i) 8–18 years of age, (ii) BMI ⩾85th percentile for age and gender based on Center for Disease Control and Prevention guidelines,[14] (iii) Latino ancestry (all four grandparents of Latino origin as determined by parental self-report) and (iv) family history of type 2 diabetes in one parent, sibling or grandparent determined by parental self-report. Participants were excluded for the following reasons: if they were taking any medication known to affect body composition, or if they had been diagnosed with a disease/s or syndrome known to affect body composition or fat distribution. SOLAR was approved by the Institutional Review Board of

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the University of Southern California. Informed written consent and assent were obtained from both parents and children before testing commenced.

### Anthropometrics and adiposity measures

A licensed pediatric health-care provider performed a detailed physical exam where Tanner staging was determined using established guidelines.[15,16] Height, weight and waist circumference (at the umbilicus) were measured to the nearest 0.1 cm, 0.1 kg and 0.1 cm, respectively. Body mass index (BMI) and BMI *Z*-scores were determined by using the EPII 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA, USA; Centers for Disease Control and Prevention[14]). Whole-body fat and soft lean tissue were measured by dual-energy X-ray absorptiometry with the use of a Hologic QDR 4500W (Hologic, Bedford, MA, USA).

### Insulin and glucose indexes

After an overnight fast, a 2-h oral glucose tolerance test was conducted with a dose of 1.75 g glucose per kg body weight (to a maximum of 75 g). Blood was sampled and assayed for glucose and insulin at − 5 min (fasting state) and 120 min (2 h) relative to glucose ingestion. Within 1 month after the OGTT visit, non-diabetic children were asked to come back to the General Clinical Research Center for an overnight visit when an FSIVGTT was performed. At time 0, glucose (25% dextrose, 0.3 g $kg^{-1}$ body weight) was administered intravenously, and insulin (0.02 U $kg^{-1}$ body weight; Humulin R (regular insulin for human injection); Eli Lilly, Indianapolis, IN, USA) was injected intravenously at 20 min. A total of 13 blood samples were collected. Plasma was collected when the FSIVGTT was performed for measuring glucose and insulin, and values were entered into the MINMOD MILLENIUM 2003 computer program (version 5.16; Richard N Bergman, University of Southern California, Los Angeles, CA, USA) for the assessment of insulin sensitivity (SI), acute insulin response (AIR) and disposition index (DI—an index of β-cell function).

### Assays

Blood samples from all time points taken during the OGTT and FSIVGTT were centrifuged (10 min, 2500 r.p.m., 8–10 °C) immediately to obtain plasma, and aliquots were frozen at − 70 °C until assayed. Glucose from the OGTT was analyzed on a Dimension Clinical Chemistry system with the use of an *in vitro* Hexokinase method (Dade Behring, Deerfield, IL, USA). Glucose from the FSIVGTT was assayed in duplicate on an analyzer (model 2700; Yellow Springs Instrument, Yellow Springs, OH, USA) using the glucose oxidase method. Insulin was assayed in duplicate by using a specific human insulin Enzyme-Linked Immunoabsorbent Assay Kit (Linco, St Charles, MO, USA). Fasting lipids ($n = 135$), including triglycerides, low-density lipoprotein (LDL)-cholesterol and high-density lipoprotein (HDL)-cholesterol, were assessed using Vitros Chemistry DT Slides (Johnson and Johnson Clinical Diagnostics Inc., Rochester, NY, USA).

### Dietary intakes

Dietary intake was assessed from two or three 24-h diet recalls using the multiple-pass technique. A bilingual dietary technician administered one recall in person during the outpatient visit with the use of three-dimensional food models. The second recall was administered by telephone by the same technician in the week after the visit. All recalls were administered or checked by a trained dietary technician in person or by telephone. Nutritional data were analyzed by using the Nutrition Data System for Research (NDS-R 2010 version 5.0_35). The Nutrition Data System for Research program calculated key dietary variables for this analysis, including mean energy, total fat, protein, carbohydrates, saturated fat, total sugar, added sugar, dietary fiber, soluble fiber and insoluble fiber. We then calculated the percent caloric intake of total fat, protein, carbohydrates, saturated fat, sugar, and added sugar and the grams of fiber per 1000 kcal. The dietary data were carefully screened for plausibility. Data were first screened by evaluating the participants' comments and no subjects were excluded; the dietary data were then examined for plausibility by performing a regression of caloric intake on BMI, and one subject was excluded because they had a standardized residual greater than 3 s.d. above the mean.

### Eating frequency analysis

The following aspects of eating frequency were examined: average number of EOs per 24 h and infrequent eating versus frequent eating. Infrequent eaters (IEs; $n = 32$) were classified as those subjects who ate less than three times per 24 h on any dietary recall, and frequent eaters (FEs; $n = 159$) were classified as those subjects who ate three or more times per 24 h on all dietary recalls. We followed a previously established eating frequency methodology[8] and did not make a distinction between meals and snacks, and instead examined eating frequency based on the number of EOs; each EO had to be at least 50 calories and at least 15 min from any previous EO.[8] Also, using methodology first published by Thompson *et al.*,[9] we calculated the eating frequency and the amount of energy consumed in three different time blocks—morning (0600–1059 hours), afternoon (1100–1659 hours) or evening/night (1700–0559 hours).

### Statistics

Data were examined for normality, and transformations were made if the data were found to be significantly different from normal. The following outcome variables were non-normally distributed and were log transformed before the analysis: protein (%kcal), 2 h glucose, fasting insulin, HOMA-IR (Homeostatic Model Assessment: Insulin Resistance), AIR, SI, disposition index, triglycerides and HDL-cholesterol, but back-transformations are displayed in the text and tables. BMI *Z*-score, carbohydrates (%kcal) and all fiber intakes were unable to be normalized within this population. A *t*-test or $\chi^2$ analyses were used to assess differences in age, sex, ethnicity and Tanner stage between eating frequency groups. Analysis of covariance analyses were used to assess differences in eating pattern and dietary intake variables, adiposity and metabolic parameters between the two eating frequency groups. In all models, the following *a priori* covariates were included: Tanner stage and sex. Mean energy intake was used as a covariate in the analysis of metabolic parameters and adiposity measures. Total fat was included as covariate for all metabolic parameters. Total fat was also included as a covariate for total lean mass and *vice versa*. Height was included as a covariate for both total fat and total lean mass. SI was included as a covariate for AIR. All analyses were performed by using SPSS version 20.0 (SPSS, Chicago, IL, USA), and the significance was set at $P \leqslant 0.05$.

## RESULTS

The basic demographic data and adiposity measures are presented in Table 1. There were 191 overweight participants who had complete anthropometric, dietary and body composition data. Metabolic outcomes were available in smaller samples. The sample was 57% male and averaged 13.5 years of age. Table 2 presents dietary data. The average number of EO per 24 h was 4.0, and 45% of the daily calories were consumed between 1100 and 1659 hours.

Table 3 presents adiposity and metabolic measures by the two eating frequency groups. We found no significant difference in sex or weight between eating frequency groups; however, we did find significant differences in age and Tanner staging. IEs tended to be older and higher in pubertal status. Independent of covariates, FEs showed a 9% lower BMI *Z*-scores ($P = 0.01$), a 9% lower waist circumference ($P \leqslant 0.01$) and a 17% lower amount of lean tissue ($P = 0.05$) compared with IEs, as well as a significantly lower BMI *Z*-score ($P \leqslant 0.01$) There were no other significant differences in adiposity measures between the two groups. There were also significant differences in metabolic outcomes, FEs compared with IEs had a 29% lower fasting insulin value ($P = 0.02$), a 31% lower HOMA-IR value ($P = 0.01$), an 11% higher AIR ($P = 0.02$), a 31% higher disposition index ($P \leqslant 0.01$) and 19% lower triglycerides ($P \leqslant 0.01$), all of which indicate that FEs had a healthier metabolic profile than IEs.

Dietary variables between IEs and FEs are depicted in Table 4. FEs ate 40% more often ($P \leqslant 0.01$) and ate 19% less per EO ($P \leqslant 0.01$), while consuming 23% or on average 431 more calories per day ($P \leqslant 0.01$) than IEs. There were no significant differences in macronutrients, fiber or sugar intake between the two groups. Additionally, FEs had significantly more EOs than IEs in the morning, afternoon and evening; however, the percentage of calories consumed during these time periods was not significantly different between the two groups.

**Table 1.** Subject characteristics[a]

| | |
|---|---|
| *Physical and adiposity measures* (n = *191*) | |
| Sex M/F | 108/83 |
| Age (years) | 13.5 ± 1.9 |
| Tanner stage | 3.3 ± 1.3 |
| Height (cm) | 159.9 ± 10.9 |
| Weight (kg) | 78.6 ± 20.5 |
| BMI (kg m$^{-2}$) | 30.3 ± 5.5 |
| BMI *Z*-score | 2.0 ± 0.5 |
| Waist circumference (cm) | 92.1 ± 12.3 |
| Total fat (kg) | 29.0 ± 10.4 |
| Total lean tissue mass (kg) | 46.2 ± 11.7 |
| Total body fat (%) | 37.1 ± 6.7 |
| *Metabolic parameters from OGTT* (n = *190*) | |
| Fasting glucose (mg dl$^{-1}$) | 91.0 ± 7.3 |
| 2 h Glucose (mg dl$^{-1}$) | 124.1 ± 19.7 |
| *Metabolic parameters from FSIVGTT* (n = *169*) | |
| Fasting glucose (mg dl$^{-1}$) | 90.7 ± 7.5 |
| Fasting insulin (μU ml$^{-1}$) | 13.7 ± 7.7 |
| HOMA-IR | 3.1 ± 1.8 |
| SI (×10$^{-4}$ min$^{-1}$ μU$^{-1}$ ml$^{-1}$)[b] | 1.8 ± 1.0 |
| AIR (μU ml$^{-1}$ × 10 min)[b] | 1466.5 ± 834.2 |
| DI (×10$^{-4}$ min$^{-1}$)[b] | 2113.8 ± 1,013.6 |
| Total cholesterol (mg dl$^{-1}$)[c] | 148.2 ± 25.7 |
| Triglyceride (mg dl$^{-1}$)[c] | 112.0 ± 55.3 |
| LDL-cholesterol (mg dl$^{-1}$)[c] | 88.1 ± 22.0 |
| HDL-cholesterol (mg dl$^{-1}$)[c] | 37.6 ± 9.6 |

Abbreviations: AIR, acute insulin response; DI, disposition Index; FSIVGTT, frequently sampled intravenous glucose tolerance test; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment-Insulin Resistance; LDL, low-density lipoprotein; OGTT, oral glucose tolerance test; SI, insulin sensitivity. [a]Data are presented as mean plus/minus s.d. [b]$n = 128$. [c]$n = 135$.

**Table 2.** Behavioral characteristics[a]

| | |
|---|---|
| *Dietary variables* (n = *191*) | |
| Eating occasions per day | 4.0 ± 1.0 |
| Energy per eating occasion (kcal) | 465.3 ± 153.8 |
| Energy (kcal) | 1792.6 ± 582.0 |
| Eating occasions in the morning | 0.9 ± 0.5 |
| % Energy consumed in the morning | 19.8 ± 14.0 |
| Eating occasions in the afternoon | 1.7 ± 0.7 |
| % Energy consumed in the afternoon | 45.2 ± 18.8 |
| Eating occasions in the evening/at night | 1.4 ± 0.8 |
| % energy consumed in the evening/at night | 35.2 ± 18.5 |
| Total fat (g per day) | 65.5 ± 26.5 |
| Total fat (%kcal) | 31.6 ± 6.1 |
| Total protein (g per day) | 68.4 ± 24.2 |
| Total protein (%kcal) | 15.9 ± 4.6 |
| Total carbohydrate (g per day) | 238.1 ± 81.8 |
| Total carbohydrate (%kcal) | 52.5 ± 7.7 |
| Total saturated fat (g per day) | 23.4 ± 9.7 |
| Total saturated fat (%kcal) | 10.9 ± 2.5 |
| Added sugars (g per day) | 66.7 ± 42.7 |
| Added sugars (%kcal) | 14.5 ± 6.9 |
| Total sugars (g per day) | 108.6 ± 49.8 |
| Total sugars (%kcal) | 24.1 ± 7.2 |
| Dietary fiber (g per day) | 14.6 ± 6.2 |
| Dietary fiber (g per 1000 kcal) | 8.4 ± 3.2 |
| Insoluble fiber (g per day) | 9.7 ± 4.5 |
| Insoluble fiber (g per 1000 kcal) | 5.6 ± 2.4 |
| Soluble fiber (g per day) | 4.2 ± 2.3 |
| Soluble fiber (g per 1000 kcal) | 2.7 ± 1.2 |

[a]Data are presented as mean plus/minus s.d.

## DISCUSSION

To our knowledge, this is the first analysis to examine the relationship between eating frequency and dietary, metabolic and adiposity measures in a sample of exclusively overweight Hispanic youth, and the first within this population to calculate the EOs and percentage of calories consumed in the morning, afternoon and evening. Hispanic youth have higher rates of obesity than non-Hispanic Whites[2] and are also at increased risk of type 2 diabetes and cardiovascular disease.[11,17,18] Our previous work has shown that frequent eating (⩾3 EOs per day) in minority youth is related to decreased visceral fat and triglycerides, despite an increased caloric intake.[7] In the present analysis, we replicated and expanded on these results in a larger and younger cohort of exclusively Hispanic youth and found that FEs compared with IEs have lower BMIs, waist circumferences, fasting insulin values, insulin resistance, triglycerides, and higher insulin responses and β-cell functioning, despite reporting consuming more calories per day.

Numerous epidemiology studies have shown that increased eating frequency is linked to decreased obesity rates,[19–23] whereas some studies have shown no or opposite relationships.[24–26] A recent longitudinal study by Ritchie,[19] with 2372 African-American and Caucasian girls (9–19 years), found that lower meal frequency was related to greater increases in BMI and waist circumference over a 10-year period, independent of socio-economic status, total energy intake and physical activity levels. Other studies have found increased eating frequency to be inversely related to waist circumferences,[19,21,27] body fat percent as measured by skinfolds,[28] and fasting glucose, insulin and lipids.[27] The current findings are similar to this and demonstrate that eating frequency is associated with lower BMI parameters and waist circumference; however, we did not see an effect on body composition as measured via dual-energy X-ray absorptiometry.

To date, our group is the first to examine the relationship between eating frequency and specific metabolic outcomes attained from an FSIVGTT. In the current analysis, we found sizable differences in insulin action, with FEs having lower fasting insulin values, insulin resistance and more robust β-cell function, elucidating that FEs have a reduced risk of type 2 diabetes and other metabolic disorders. A cross-sectional analysis performed by Smith *et al.*[27] in a mixed population of 2775 young adults (26–36 years) found increased eating frequency to be negatively associated with fasting glucose and insulin, triglycerides, total cholesterol and LDL cholesterol. However, these findings were only significant in men, and no explanation of exclusion factors was included. In a crossover controlled feeding trial by Leidy *et al.*,[29] with 13 overweight or obese males, frequent eating (6 EOs) vs less frequent eating (3 EOs) resulted in a 4% decrease in fasting glucose and a 20% decrease in fasting insulin, across the 11-h testing period. However, overall there is a paucity of data linking eating frequency to specific metabolic disease markers and more research is warranted, especially in high-risk populations, such as Hispanic youth.

There are numerous potential mechanisms to explain our findings. Several experimental studies in which the eating frequency groups were designed to be eucaloric (i.e., calories to support weight maintenance) or isocaloric (i.e., matched on caloric intake) found that frequent eating was linked to decreases in hunger[30,31] and increased satiety responses.[32] However, the previous study by Leidy *et al.*[29] found increased EOs led to higher satiety throughout the day, and also higher premeal hunger ratings.[29] Thus, in a free-living environment, it is foreseeable that more EOs may lead to more total calories throughout the day, but possibly less calories consumed per EO. National adult studies support this finding and have shown that increased EOs results in increased daily quantity of foods/beverages consumed, as well as daily energy density (kcal g$^{-1}$) of all foods and beverages

**Table 3.** Adiposity measures and metabolic parameters between eating frequency groups[a,b]

| | *Infrequent eaters* (n = *32*) | *Frequent eaters* (n = *159*) | P-*value* |
|---|---|---|---|
| *Physical and adiposity measures* (n = *191*) | | | |
| Sex M/F | 23/9 | 85/74 | 0.06 |
| Age (years) | 14.4 ± 1.7 | 13.3 ± 1.9 | ⩽0.01 |
| Tanner stage | 4.0 ± 1.2 | 3.2 ± 1.3 | 0.02 |
| Weight (kg) | 90.8 ± 8.0 | 90.0 ± 7.3 | 0.09 |
| BMI *Z*-score | 2.2 ± 0.3 | 2.0 ± 0.5 | ⩽0.01 |
| Waist circumference (cm) | 99.6 ± 10.7 | 90.6 ± 12.1 | ⩽0.01 |
| Total fat (kg) | 33.1 ± 10.5 | 28.2 ± 10.3 | 0.61 |
| Total lean tissue mass (kg) | 54.0 ± 9.9 | 44.6 ± 11.4 | 0.05 |
| Total body fat (%) | 36.6 ± 7.9 | 37.2 ± 6.4 | 0.25 |
| *Metabolic parameters from OGTT* (n = *190*) | | | |
| Fasting glucose (mg dl$^{-1}$) | 93.3 ± 9.0 | 90.5 ± 6.9 | 0.44 |
| 2 h Glucose (mg dl$^{-1}$) | 126.3 ± 18.5 | 123.7 ± 20.0 | 0.50 |
| *Metabolic Parameters from FSIVGTT* (n = *169*) | | | |
| Fasting glucose (mg dl$^{-1}$) | 94.0 ± 7.9 | 90.0 ± 7.3 | 0.20 |
| Fasting insulin (μU ml$^{-1}$) | 18.0 ± 10.2 | 12.7 ± 6.8 | 0.02 |
| HOMA-IR | 4.2 ± 2.5 | 2.9 ± 1.6 | 0.01 |
| SI (×10$^{-4}$ min$^{-1}$ μU$^{-1}$ ml$^{-1}$)[c] | 1.5 ± 1.2 | 1.8 ± 1.0 | 0.78 |
| AIR (μU ml$^{-1}$ × 10 min)[c] | 1304.1 ± 902.6 | 1466.5 ± 819.2 | 0.02 |
| DI (×10$^{-4}$ min$^{-1}$)[d] | 1463.7 ± 830.4 | 2127.0 ± 938.9 | 0.01 |
| Total cholesterol (mg dl$^{-1}$)[e] | 144.7 ± 20.3 | 149.0 ± 26.8 | 0.72 |
| Triglycerides (mg dl$^{-1}$)[e] | 132.3 ± 51.7 | 107.2 ± 55.3 | ⩽0.01 |
| LDL-cholesterol (mg dl$^{-1}$)[e] | 84.1 ± 14.9 | 89.1 ± 23.3 | 0.75 |
| HDL-cholesterol (mg dl$^{-1}$)[e] | 34.1 ± 6.7 | 38.5 ± 10.0 | 0.28 |

Abbreviations: AIR, acute insulin response; ANCOVA, analysis of covariance; DI, disposition index, FSIVGTT, frequently sampled intravenous glucose tolerance test; HDL, high-density lipoprotein; HOMA-IR, Homeostatic Model Assessment-Insulin Resistance; LDL, low-density lipoprotein; OGTT, oral glucose tolerance test; SI, insulin sensitivity. [a]Data are presented as mean plus/minus s.d. [b]A *t*-test (for continuous variables) and $\chi^2$ analysis (for categorical variables) assessed differences in age, sex and Tanner stage between groups. [c]ANCOVA analysis of adiposity measures and metabolic parameters between infrequent eaters and normal/frequent eaters. *A priori* covariates included: Tanner stage, sex, mean energy (for adiposity and metabolic parameters), total fat (for metabolic parameters), total fat and height (for total lean), total lean and height (for total fat) and SI (for AIR); $n = 128$. [d]$n = 124$ (four outliers were omitted). [e]$n = 135$.

**Table 4.** Dietary characteristics between eating frequency groups[a,b]

| | *Infrequent eaters* (n = *32*) | *Frequent eaters* (n = *159*) | P-*value* |
|---|---|---|---|
| *Dietary variables* (n = *191*) | | | |
| Eating occasions per day | 2.6 ± 0.6 | 4.3 ± 0.8 | ⩽0.01 |
| Energy per eating occasion (kcal) | 553.9 ± 177.6 | 447.4 ± 142.7 | ⩽0.01 |
| Energy (kcal per day) | 1434.0 ± 523.8 | 1864.8 ± 567.6 | ⩽0.01 |
| Eating occasions in the morning | 0.6 ± 0.5 | 0.9 ± 0.5 | ⩽0.01 |
| % energy consumed in the morning | 19.5 ± 20.9 | 19.9 ± 12.3 | 0.88 |
| Eating occasions in the afternoon | 1.1 ± 0.5 | 1.8 ± 0.7 | ⩽0.01 |
| % energy consumed in the afternoon | 45.3 ± 20.6 | 45.2 ± 18.4 | 0.97 |
| Eating occasions in the evening/at night | 0.9 ± 0.4 | 1.4 ± 0.6 | ⩽0.01 |
| % energy consumed in the evening/at night | 37.5 ± 20.6 | 34.7 ± 18.1 | 0.43 |
| Total fat (%kcal) | 32.4 ± 6.9 | 31.4 ± 5.9 | 0.44 |
| Total protein (%kcal) | 17.1 ± 5.4 | 15.6 ± 4.4 | 0.16 |
| Total carbohydrates (%kcal) | 50.4 ± 9.3 | 52.9 ± 7.4 | 0.14 |
| Total saturated fat (%kcal) | 11.5 ± 2.9 | 10.7 ± 2.4 | 0.15 |
| Total sugars (%kcal) | 21.9 ± 8.1 | 24.6 ± 7.0 | 0.07 |
| Added sugars (%kcal) | 13.2 ± 7.1 | 14.7 ± 6.9 | 0.15 |
| Dietary fiber (g per 1000 kcal) | 8.5 ± 3.9 | 8.4 ± 3.1 | 0.76 |
| Insoluble fiber (g per 1000 kcal) | 5.7 ± 2.9 | 5.6 ± 2.3 | 0.65 |
| Soluble fiber (g per 1000 kcal) | 2.7 ± 1.5 | 2.7 ± 1.1 | 0.97 |

Abbreviations: ANCOVA, analysis of covariance. *A priori* covariates used: Tanner stage and sex. [a]Data are presented as mean plus/minus s.d. [b]ANCOVA analysis of dietary variables between infrequent eaters and normal eaters.

reported.[33] One national adult study[6] showed increased daily eating frequency to be associated with increased daily energy intake, where participants who consumed ⩾5 EOs per day consumed approximately 800 kcal per day more than those who consumed ⩽2 EOs per day. In free-living populations, research consistently shows a positive relationship between eating frequency and caloric intake, yet also is more regularly inversely related to obesity and adiposity measures, thus challenging the energy balance theory. In the present analysis, FEs ate more often than IEs throughout the day, including morning, afternoon and evening, revealing that the current finding is not reflective of IEs skipping any one meal. These data provide evidence that the

particular meal skipped may be less relevant to adiposity than eating less than three EOs per day. Therefore, analyses that only look at skipping any one particular meal, such as breakfast, may not be gleaning the entire story of how eating patterns relate to health outcomes. Given the irregular eating patterns of Hispanic youth, more research is warranted examining how the number of EOs per day affects energy intake, satiety/hunger measures and subsequent adiposity and metabolic parameters.

It is likely that eating frequency impacts obesity and related metabolic diseases through a combination of different mechanisms. For decades, the public and media outlets have advertised and promoted more frequent EOs as a means to increase one's metabolism and optimize weight loss. Yet, research conducted in whole-room calorimetry chambers has consistently shown that eating frequency is not associated with changes in the thermic effect of food, basal metabolic rates or 24-h energy expenditure.[34,35] However, in the present study, we did find numerous relationships between eating frequency and metabolic outcomes. The current analysis showed that eating more often is linked to lower fasting insulin values, as well as to decreased insulin resistance, higher insulin secretion rates and improved β-cell functioning. Elevated HOMA-IR values, fasting insulin and insulin resistance have been shown to elevate obesity risk in youth populations,[36] but further research in this area is needed.

Another possible mechanism involves lipid metabolism. Our study found that FEs had lower circulating triglycerides than IEs. IEs showed an increased caloric intake per EO, and binge eating behaviors have been previously linked to increased triglycerides.[37] It is also possible that FEs have less visceral fat. The accumulation of visceral fat has been positively associated with fasting insulin and triglycerides.[7,38] It is also hypothesized that visceral fat increases hepatic portal free fatty acid concentrations, which in turn are stored as triglycerides, stimulate hepatic gluconeogenesis and hinder hepatic clearance of insulin, thus promoting a vicious cycle of hyperinsulinemia, elevated plasma glucose concentrations and dyslipidemia.[39] We did not have enough subjects with magnetic resonance imaging data to include visceral fat as a measure within this analysis, but did see both reduced visceral fat and triglycerides among FEs in our previous analysis of minority youth.[7] More research, especially randomized controlled feeding trials analyzing the possible relationship between visceral adiposity, triglycerides, eating frequency and metabolic disease risk is merited.

There are several limitations to consider in the present study. Eating frequency has been found to be positively related to physical activity,[28,40] and some studies have shown this to have a mitigating effect on the relationship between eating frequency and adiposity,[40] while others have not.[19,28] The SOLAR study did not collect physical activity data; however, our previous work did not find an association between eating frequency and physical activity (measured by accelerometry) in overweight minority youth. Another limitation is possible under-reporting, especially by overweight/obese participants.[41,42] However, this population is very homogenous, with 79% being obese, and thus under-reporting would be expected to be consistent throughout.[43] Furthermore, we used multiple exclusion criteria to assess implausible reporters, including subject comments, as well as running a regression with caloric intake and BMI *Z*-scores and excluding subjects who were $>$ or $<3$ s.d. from the mean. SOLAR is a longitudinal trial, yet the number of participants with complete dietary and body composition data at multiple time points was too low to conduct mixed modeling, thus driving us to the current cross-sectional analysis. This sample also had a relatively small number of IEs, yet the percentage of the sample is consistent with our previous work[7] and others.[44] Owing to the cross-sectional nature of this data set inferring a causal pathway is difficult. However, these results replicate our previous findings and because this is a youth population, we expect that the eating frequency patterns are, in fact, having a role in promoting obesity and metabolic diseases. These results highlight the need for more intervention work in this area to truly assess causality.

In summary, eating three or more times per day is associated with healthier outcomes for obesity and metabolic risk, despite being linked to increased reported daily energy intake. Given that Hispanic youth are at such a high risk of obesity and associated metabolic disorders, it is important to identify nutrition behaviors that may potentially reduce this risk. These results as well as our group's and other's previous findings support that intervention work is needed to investigate the potential causal mechanism of how eating frequency affects obesity and metabolic disease risk, particularly in Hispanic youth.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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